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VOL. LX DECEMBER, 1950. No. 12

CLOSURE AND REVISION FOLLOWING THE FENESTRATION OPERATION.*

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INTRODUCTION.

The two chief problems in the prognosis of the fenestration operation for clinical otosclerosis are: first, what will be the immediate gain for speech, and second, will the improvement be permanent. The first question we have analyzed recently[1] on the basis of a long-term study of the patients fenestrated by one of us (T. E. W.) at McMillan Hospital (Washington University). The present study deals with the second question and reports the incidence of closure in the same series of operations; it also includes our very disappointing experience with the "revision" operation in which the surgeon reopens a fenestra that has been closed by the regrowth of bone.

SURGICAL PROCEDURES.

Before October 23, 1946, the cartilage stopple[2] was routinely employed in the fenestration operation, following the

*This study was begun under Contract N6onr-272 between the Office of Naval Research and Central Institute for the Deaf. It was continued under Contract V1001M-577 between Veterans Administration and Central Institute for the Deaf. The routine testing and the collection of data were later transferred to the Hofheimer Audiology Laboratory at McMillan Hospital. The work there is supported by a grant from the Hofheimer Foundation.

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mistaken idea that it tended to prevent the normal regrowth of bone and consequent closure. When it became apparent that this was emphatically not the case[4] the stopple was abandoned. The cases in which the stopple was employed are not included in the present study, but a few early cases in which the stopple was omitted are fortunately available now (April, 1950), four or five years after operation.

Although there have been modifications from time to time in the incisions, in the extent and technique of mastoidectomy and in the pre- and post-operative care of the patient, the technique for creation of the fenestra has been constant. Until March 14, 1947 (Case No. 185), a Cameron loupe was used for magnification. From Case No. 185 to date a 6× microscope has been used with constant irrigation and suction as described by Shambaugh.

Starting with Case No. 222 the size of the fenestra has been measured and noted. It varies between 4.5 and 2.25 mm. in length by 1.0 - 1.25 mm. in width; the average size being 3.25 × 1 mm.

In all cases, the fenestra has been created with a polishing burr. The edges have been cleaned with dental picks and a gold burnishing "burr" used to eburnate the bone. Utmost care has been exercised in obtaining complete hemostasis and in insuring against bone dust in the fenestra.

SELECTION OF CASES AND TESTS FOR CLOSURE.

The present analysis is based on 170 cases (through Case No. 314) operated (without stopple) before July 1, 1949. Of these, 153 were operated since October 22, 1946, when the last stopple operation was performed. They are, therefore, consecutive cases except that we have been forced to omit 15 cases on whom we have been unable to perform the necessary follow-up tests.

The routine tests of hearing included "loss for speech" measured by P.A.L. Auditory Test No. 9 (spondees), "average loss for the speech range" (measured as the average pure

tone loss at frequencies 512, 1024, 2048 and 2896 cycles per second), and the fistula test for patency of the fenestra in doubtful cases. The tests of hearing have been fully described elsewhere.[3,4]

The first complete post-operative tests were usually done during the second month after operation. The plan of follow-up called for another complete test at six months, one at a year, and then at annual intervals thereafter. For practical reasons this schedule could not be followed very closely. Some patients were tested more frequently, particularly when they complained that they were losing their hearing again. Actually, for the group as a whole, the tests were scattered nearly at random except for a gradual decrease in frequency over the first year and a half. The annual testing thereafter has been a little more regular.

In the 170 fenestration operations there were 20 that we have classed as failures because they did not show as much as 10 db gain for speech at the first complete post-operative test. The causes of these "immediate failures" have already been analyzed[1] but it is worth recalling that the incidence of such "failure," reckoned in the basis of all (non-stopple) original fenestrations including those not followed up, is 20/185 — 10.81 per cent. Because many (13) "failures" did not return for follow-up, we have omitted from our calculations all "failure" because of very early closure. In other words, we reckon the incidence of closure on the basis of the "initially successful" operations that gave at least 10 db gain for speech.

CLOSURE.

Closure of the fenestra is suspected when any follow-up test shows that the patient's threshold for speech is 10 db or more higher than it was at the first post-operative test. Closure is proved by a negative fistula test, *i.e.* no nystagmus or vertigo when gentle pressure is applied to the skin graft over the fenestra. Furthermore, the diagnosis of closure was confirmed at operation in the 14 cases on whom "revision" was attempted. One case (No. 5) showed a dense fibrous scar in the fenestra. All of the 13 others were true bony closures.

Only the cases proved closed by the fistula test (when it was clear) are counted in the calculations below, although a few others suffered more than 10 db loss of their initial gains for other reasons.[1]

Two cases are complicated, one by injury from an explosion just prior to "closure" and the second by some nerve deafness and rather serious psychological difficulties. In these two the evidence for closure is doubtful or incomplete. We arbitrarily include the former (No. 270) but reject the latter. This probably gives the fairest overall figure for incidence of closure.

We have in all, then, 21 definite and one presumed closures. This is 14.6 per cent of the 151 initially successful operations (without stopple) on whom we have adequate follow-up data. The percentage may still increase slightly as 15 cases are still in the second six month period after the operation when closure is still fairly frequent.

When closure occurred it occurred within the first year and often within the first six months after operation. We have tests on 73 cases after 1½ years or more (9 of them 3½ years or more) and not one has closed later than 12½ months after operation (see Figure 1).

The median of the intervals at which the 22 closures were last proved to be still open is 2.7 months. The median of the intervals at which they were first found to be closed was 7.5 months. The earliest closure was within 1 month. Only one case that is known to have been open at 9 months subsequently closed. The most probable time of closure, if it is to occur, is, therefore, during the fifth month after operation. Figure 1 shows that this figure has not altered significantly since the beginning of our series. Furthermore the incidence of closure seems to be constant. Our three successive groups of 50 cases each contain 8, 7 and 7 closures respectively.

The 132 successful cases that have remained open show a slight mean loss in hearing for speech (based on Test No. 9) since the first post-operative test: —2.77 db ± 4.7 db. The distribution of the losses about the mean is not skewed. This

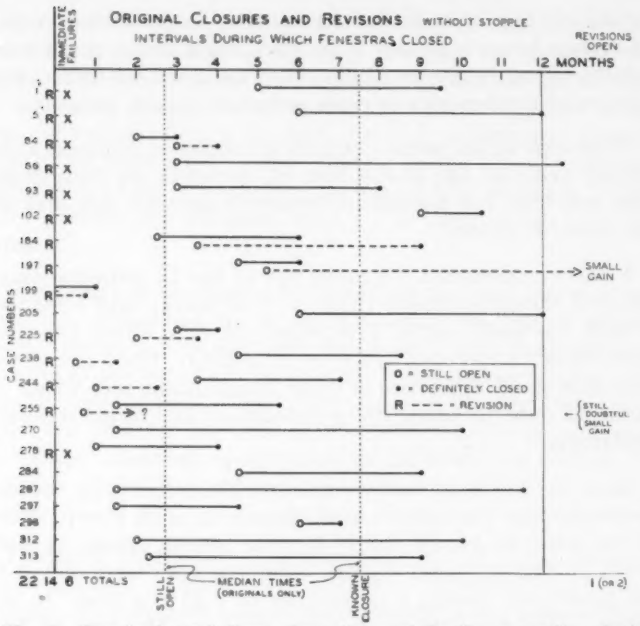

Fig. 1. The horizontal lines show the period after operation during which the closure took place. Each open circle indicates the last test at which hearing was still up to or within 10 db of the immediate post-operative level. The filled circle indicates the date of the first test at which there was clear evidence that closure had occurred. All intermediate tests in which the evidence was equivocal are omitted.

slight but significant loss of sensitivity does not seem to increase after the first year and apparently not after the first six months, but more cases are needed to confirm these latter impressions. This slight late loss of nearly 3 db should be added to the expected minimum residual loss of 20 db that we discussed in a previous paper.[1] The late loss may be due to a stiffening of the skin graft and other tissues as scar tissue becomes mature. Further details concerning changes in auditory sensitivity during the first year after fenestration are reserved for later publication.

CLOSURE IN RELATION TO AGE.

Closure is obviously due to a normal healing process. It seems to be the general opinion that young patients (in their

teens) are less suitable for fenestration because their repair of broken bones is known to be more rapid and perhaps more effective than repair in adults. It is assumed, therefore, that closure of the fenestra is more probable in such patients.

Inasmuch as our series contains a number of patients under twenty years of age at the time of operation we have examined our data for possible correlations between age and the incidence of closure.*

First we calculated the mean age of the 23 patients whose original fenestras closed (31.0 ± 11.9 years) and compared it with the mean age of 170† other patients whose fenestras were known to be open (33.6 ± 9.0 years). The t-test shows that this small difference is quite insignificant and would be expected once in three times because of the large standard deviations.

As a more delicate test we grouped the patients by decades and compared the incidence of closure in each group. Four of our closures are in the 10-19 year group, giving an incidence of 33.3 per cent. On the other hand we have only 5 in the 30-39 year group, a percentage of 7.25 per cent. In the entire group from 20 to 69 years we have 19 cases or 10.5 per cent. Further calculations show that the difference between the 10-19 and the 30-39 year groups is significant at about the .02 level. The difference between the 10-19 year and the 20-69 year groups is also significant (at about the .04 level). All of the other differences would be expected to occur by chance oftener than once in twenty times. The data and calculations thus support the general impression that a fenestra in a teen-age patient is more likely to close than one in an adult. Because of the small numbers of cases involved we do not regard our data on this point as definitive, but they are certainly very suggestive.

*The calculations below were made in April, 1950, before the final classification of several doubtful cases had been settled. On the basis of more recent data two cases (aged 28 and 31) were omitted from and one case (age 39) was included in the final series discussed in the previous section as compared with the group here analyzed for the effect of age. These shifts do not significantly affect the results to be presented.

†The series is larger than the series analyzed in the previous section as cases through No. 332 and some additional follow-ups on earlier cases were available.

A very important detail of the relation of closure to age, however, is found among the very youngest members of the series. *The fenestras of the three youngest, aged 10, 12 and 13 years, all closed.* The next six, aged 14 to 18 years inclusive, all remained open. In fact, the high incidence of closure among the teen-agers is due entirely to the three youngest. Otherwise, except possibly for the rather low incidence in the third decade, the closures seem to be distributed quite at random.

Our practical conclusion is, therefore, that the fenestration operation should not be performed before the age of fifteen years. Patients younger than this should wear hearing aids, in spite of the social advantage that would follow from a successful fenestration. This point becomes doubly important when we consider the very unfavorable outcome of attempts to "revise" closures, as described in the following section.

REVISION.

Fourteen of the 22 fenestras that closed were revised. Unfortunately revision was uniformly unsuccessful,* either because of immediate failure (particularly in the cases revised in 1946) or because, with one or possibly two exceptions, the fenestra closed a second time (see Figure 1). Only these 2 out of the 14 revisions can be considered even partially successful, with gains of 8 db and 14 db (equivocal, based on contradictory data) respectively. In addition one other case that closed has nevertheless improved by 6 db. All of the 11 others were made worse by the revision. Six ears were totally deafened at the operation, most of them by tearing the membranous labyrinth that had adhered to the new bone. The failures are easy to understand. The scar tissue makes the

*In addition to the series here presented, one of us (T. E. W.) has also revised 61 fenestrations in which he employed the stopple cartilage. Adopting the same criterion of success, i.e. a gain for speech of 10 db or more, and classing as doubtful those on whom the data is insufficient or equivocal, the results are a little better than in the present series, i.e.

Successful:	28 per cent
Failure, including closures:	65 per cent
Doubtful:	7 per cent

If the final result is compared to the status before the **original** operation the percentage of "successful" cases becomes 38 per cent. The percentage of original closures was far higher with the stopple than in the present series since the stopple was abandoned.

operation far more difficult surgically, and five of the immediate failures are among our first six revisions.

The extraordinarily high percentage of closures of the revised fenestras raises a question as to whether the second closure is due in some way to the previous operation. Does the old scar tissue favor closure? Have the tissues been "stimulated" in some way to more active repair? Does the original closure automatically select the cases with the strongest constitutional tendency to form new bone and close the fenestra? Against the idea that the previous operation favors closure is the rather scanty data in Figure 1. Here it is clear that on the average each ear closed the second time after about the same length of time that it did the first time. Even if closure has been made more likely it has not been made much more rapid. The "constitutional" theory will be tested when we have accumulated enough cases in which the opposite ear is fenestrated. This now seems to be the operation of choice in case of "failure." In any case we have abandoned the classical "revision operation" because of the very unfavorable results here reported.

SUMMARY.

Follow-up tests of hearing and for closure of the fenestra have been conducted over periods from 6 months to 5 years on 170 patients.

Closure of the fenestra occurred in 22 cases or 14.6 per cent of the 151 initially successful cases. Closure occurred in all three of our patients who were less than 15 years old, but there is only a slight relation if any to age from 15 years onward.

Closure usually occurred between 2.7 and 7.5 months after operation. Only one of the 22 closed after more than 9 months and none after one year.

The cases that did not close show a mean loss of hearing for speech of 2.77 $\pm$ 4.7 db that occurred during the first year after operation but (on the average) did not increase further.

The operation was "revised" in 14 cases of closure. The results were very unsatisfactory. Six cases were immediate failures and six more closed again after about the same interval as after the original operation. Only one revised fenestra is apparently open after one year and the gain is only 8 db. We have abandoned the revision operation. There has been no apparent relation between closure and technique.

REFERENCES.

1. Davis, H., and Walsh, T. E.: The Limits of Improvement of Hearing Following the Fenestration Operation. The Laryngoscope, 1950, 60, 273-295.

2. Lempert, J.: Fenestra Nov-Ovalis with Mobile Stopple. *Archives of Otolaryngology*, 1945, 41, 1-41.

3. Thurlow, W. R., Davis, H., Silverman, S. R., and Walsh, T. E.: Further Statistical Study of Auditory Tests in Relation to the Fenestration Operation. The Laryngoscope, 1949, 59, 113-129.

4. Thurlow, W. R., Silverman, S. R., Davis, H., and Walsh, T. E.: A Statistical Study of Auditory Tests in Relation to the Fenestration Operation. The Laryngoscope, 1948, 58, 43-66.

INTRALARYNGEAL CORRECTION OF BILATERAL ABDUCTOR PARALYSIS: A MODIFICATION OF THE THORNELL OPERATION.*†

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The increase of goiter surgery in the past 50 years has caused a corresponding rise in the number of vocal cord paralyses. Trauma of the recurrent laryngeal nerves at the time of surgery or during the postoperative period has produced numerous instances of bilateral abductor paralysis with its crippling and depressing effects on the unfortunate victims; yet it has been only during the past decade that methods have been devised for the successful correction of this dread complication.

The ideal solution of the problem would, of course, be the restoration of function for the paralyzed posterior cricoarytenoid muscles and a return to the normal physiology of the larynx; however, all attempts to reach this goal, whether by nerve anastomosis or by muscle transposition, have ended in failure. Other efforts have been directed toward widening of the glottic opening to establish an adequate airway without sacrificing the voice of the patient.

Early attempts to achieve these results by intralaryngeal cordectomy or ventriculocordectomy failed, due to the development of postoperative stenoses of the larynx. An arytenoidectomy was first performed by veterinary surgeons for relief of dyspnea in roarer horses, due to recurrent nerve

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paralysis. A thorough survey of the literature reveals one succcessful case of arytenoidectomy by laryngofissure reported by Baker[1] in 1916; however, his work was passed over, and, in 1932, Hoover[2] followed the example of Killian, Dahmann and Albrecht by performing several satisfactory submucous resections of one vocal cord. Other surgeons failed to duplicate these results and, in 1936, Loré[3] suggested a more extensive submucous resection of one vocal cord with complete removal of the arytenoid cartilage. Unfortunately, all of those operations resulted in partial or complete loss of the voice.

In 1939, King[4,5] described his famous operation for the surgical treatment of bilateral abductor paralysis and stimulated new interest in this field. Originally he conceived the idea of transposing the anterior belly of the omohyoid muscle to the arytenoid cartilage, but he[6] soon discovered that his best results were obtained in those cases in which the arytenoid cartilage was disarticulated and fixed in lateral displacement together with the corresponding vocal cord. His success in widening the airway while preserving the voice encouraged many others to follow his example, and his technique has been employed with good results by Morrison[7] and others.

In the following year, 1940, Kelly[8,9] described a procedure, in which arytenoidectomy was carried out through a small window in the thyroid cartilage. Great interest greeted this new method, and several modifications were introduced in an attempt to perfect it. Galloway[10] suggested mobilization of the arytenoid cartilage and its fixation to the extralaryngeal structures outside the thyroid window. A similar method of transfixing the arytenoid cartilage was described by McCall and Gardiner.[11] Wright[12] sutured the extreme posterior end of the vocal cord to the perichondrium on the outer surface of the thyroid cartilage. Orton[13,14] used Trotter's[15] lateral transthyroid approach, and Woodman[16] removed a portion of the thyroid ala to obtain better exposure.

While all of these techniques were successful in a large number of cases, Kelly[17] himself was the first to admit the difficulties encountered in the extralaryngeal approach. In an

excellent summary written in 1944, he calls attention to the difficulty in exposure, to the elusion of the arytenoid cartilage, to the breaking of sutures on coughing or vomiting, to possible lacerations through the laryngeal mucosa, and to the breakdown of cartilage in elderly patients, with subsequent delay in healing.

Two years ago, in an effort to overcome those difficulties, William Thornell,[18] of Cincinnati, described a new intralaryngeal approach to the surgical correction of bilateral vocal cord paralysis. The Thornell operation is founded on the use of the Lynch suspension laryngoscope and consists of complete intralaryngeal arytenoidectomy with lateral fixation of the corresponding vocal cord by electrocautery.

Since this first report 22 such operations have been successfully carried out, and several modifications have been introduced. In each case, the patient has been observed for a period of several months after surgery to permit an accurate appraisal of the functional result. Thornell[19-21] has performed seven of those operations and has added the use of an O'Dwyer intubation tube and an acrylic obturator. Havens[22] and his colleagues at the Mayo Clinic have performed 11 successful intralaryngeal arytenoidectomies and are very pleased about their results. Edward King,[23] of Los Angeles, has used this procedure twice, and I am reporting two cases that I operated upon last year. These two patients had developed bilateral vocal cord paralysis as a result of thyroid surgery. In both cases I employed a modification of the original Thornell operation, and obtained excellent results with this technique.

OPERATIVE TECHNIQUE.

In place of the cumbersome suspension apparatus, Seiffert's self-retaining laryngoscope (see Fig. 1) was used for exposure of the operative field; the head of the patient was supported on a mechanical head rest. Preoperative medication consisted of nembutal (1.5 gr.) at bedtime and morphine (one-eighth gr.) and scopolamine (1/200 gr.) one hour before surgery. Intravenous injection of thiopental sodium (U.S.P.)

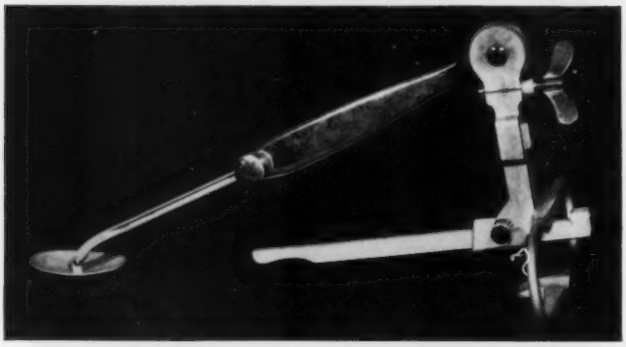

Fig. 1. Seiffert's self-retaining laryngoscope.

was supplemented by inhalation anesthesia of nitrous oxide and oxygen through the tracheotomy cannula. The operative area was further anesthetized by the local application of 2 per cent pontocaine.

After abolition of the pharyngeal reflexes and complete relaxation of the cervical muscles had been obtained, Seiffert's self-retaining laryngoscope was introduced and the larynx was exposed. An incision, 1 cm. in length, was then made over the superior surface of the arytenoid cartilage and extended anterolaterally into the aryepiglottic fold, as described by Thornell (see Fig. 2). The wound edges were separated, and the arytenoid cartilage was identified. It was stripped of its various muscular attachments by sharp submucous dissection, and disarticulated at the cricoarytenoid joint. In addition to the Lynch operating instruments, two very sharp knives, a straight bistoury and an angular bistoury with double edge blades (see Fig. 3), were adapted by me for this part of the procedure, because it is extremely important to avoid injury of the mucous membrane on the lateral wall of the larynx.

Following complete removal of the arytenoid cartilage a guarded curved electrocautery tip was inserted deeply through the incision along the course of the thyroarytenoid muscle, beneath the vocal cord and into the space previously occupied

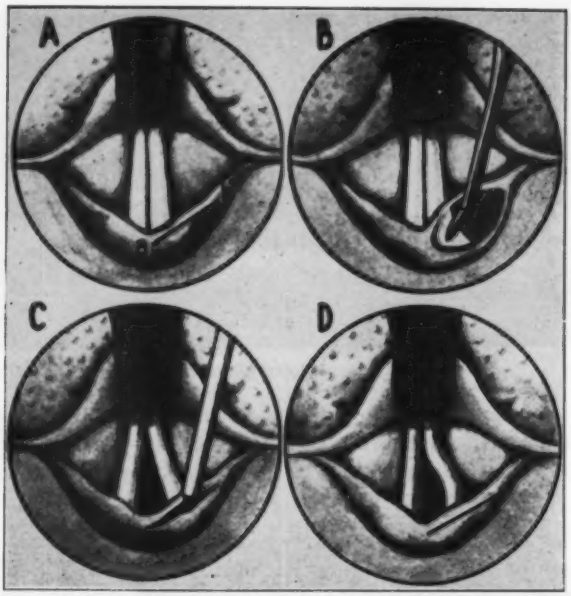

Fig. 2. Schema showing (A) the incision (a-b) over the arytenoid area extending into the aryepiglottic fold; (B) position of the arytenoid cartilage, tightly held by a grasping forceps; (C) lateral displacement resulting from the removal of the arytenoid cartilage, with the guarded electrocautery point in position for cauterization; (D) lateral displacement of the posterior two-thirds of the vocal cord. (Reproduced from the Archives of Otolaryngology, by courtesy of Dr. William C. Thornell.)

by the arytenoid cartilage. Further lateral fixation of the posterior half of the vocal cord is thus obtained by the resulting contraction from the electrocautery. A small amount of adrenalin (1 to 10,000 solution) was used for local infiltration in one of the cases, but bleeding was minimal in both operations and readily controlled with the electrocautery unit. The incision was allowed to remain open to afford drainage.

An acrylic obturator was then inserted between the cords (see Fig. 4); it was anchored inferiorly to the flange of the tracheal cannula and superiorly to the skin of the cheek. This obturator is tolerated well and causes surprisingly little reaction to the soft tissues of the larynx. I feel that its use is particularly important in cases where a previous laryngeal

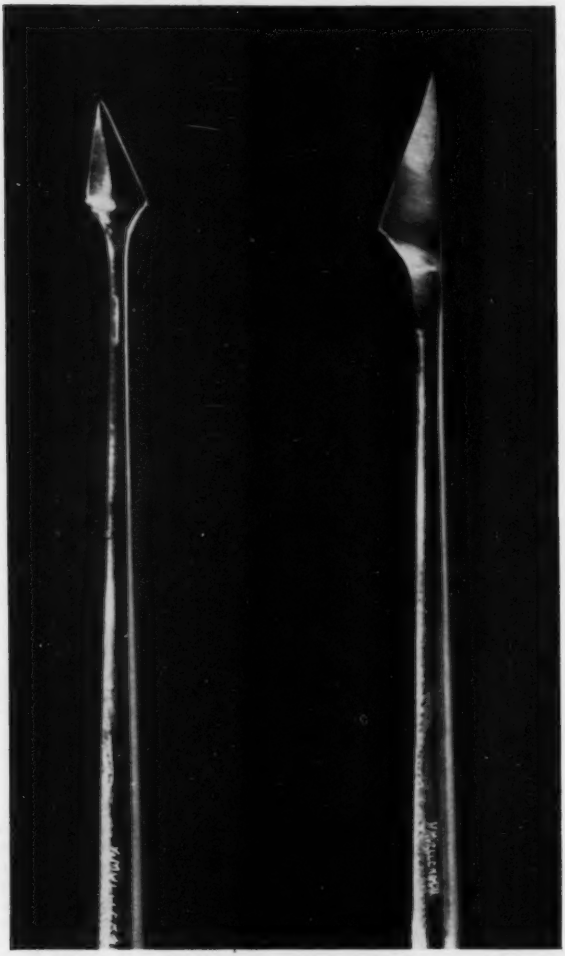

Fig. 3. Straight and angular bistoury knives mounted on long laryngeal handles.

procedure has been carried out with subsequent scarring of the laryngeal structures. One of my cases falls within this group; it is, I believe, the only case on record in which a suc-

cessful intralaryngeal arytenoidectomy has been performed on the same side as an unsuccessful extralaryngeal operation.

Fig. 4. Acrylic obturator.

CASE REPORTS.

Case 1: The patient, a 48-year-old white woman, was seen at St. Francis Hospital on Feb. 26, 1949. She presented a history of four thyroidectomies over a period of 28 years. Following the last operation in May 1948, she had noticed hoarseness, and in October 1948, respiratory difficulties developed. Her symptoms had become progressively worse since that time, forcing her to spend most of the time in bed.

At the time of my first examination, the patient was sitting up in bed, in acute respiratory distress, with marked dyspnea and a very loud laryngeal stridor; she was very nervous and jittery. The inspiratory crow was so noisy that it could be heard at the other end of the hall, in spite of closed doors. Indirect laryngoscopy revealed clockwise rotation of the entire larynx, with fixation of both vocal cords in the midline. The remainder of the ear, nose and throat examination revealed extensive scarring of the soft tissues of the neck.

An emergency tracheotomy was carried out, followed by immediate relief in breathing. During the convalescent period the patient developed a lobar pneumonia, an acute otitis media, and right heart failure, and her release from the hospital was delayed until March 17. Seven weeks later, on May 5, she was again admitted to the hospital complaining of nervousness, shortness of breath, pain in the left chest, and swelling of the lower extremities of four days' duration. The diagnosis on this admission was hypothyroidism and hypoparathyroidism with cardiac decompensation, and secondary bronchopneumonia. Under indicated therapy she improved very slowly until her discharge from the hospital on Aug. 13. She continued under strict medical observation, until her general condition had returned to normal and she was pronounced ready for intralaryngeal surgery.

On Dec. 18, 1949, the patient re-entered St. Francis Hospital, and on the following day a left arytenoidectomy was carried out through Seiffert's self-retaining laryngoscope, as outlined above. The left side was chosen for this procedure, because vibratory excursions of the right cord were more pronounced, although the rotation of the larynx made an operation on this side technically more difficult.

The postoperative course was uneventful, except for slight fever and increased pulse rate during the first three days. The acrylic obturator was removed on the fourth day. The moderate postoperative edema subsided quickly. The patient was discharged on the eighth postoperative day. She was again hospitalized on Jan. 24, 1950, when the tracheotomy stoma was closed under local anesthesia.

Since the operation the patient has lost her nervousness and all difficulties in breathing. She is taking care of her home and fulfills all the duties of housewife, though she has been advised by her cardiologist to avoid unnecessary strain. Her voice is clearer than it was at my first examination, although somewhat weaker.

Case 2: The patient, a 52-year-old white woman, was first seen in my office on May 31, 1949. She had had a thyroidectomy in July, 1948. Three months later she had noticed slight difficulty in breathing; this had become progressively more severe, until she had been forced to give up her position as a secretary and refrain from any and all exercise. She also complained of severe nervousness, loss of 40 pounds in weight, and stiffness of the neck, apparently due to muscular spasm from keeping the head tilted backwards in the position of best respiration.

On laryngeal examination, both vocal cords were found to be fixed in the midline, with only a 1 mm. glottic opening present. A low transverse cervical thyroidectomy scar was noted. The patient was thin, extremely nervous and very worried. She had an inspiratory stridor, which became pronounced with slight exercise or emotional changes. A general medical examination by an internist revealed hypertensive heart disease and a severe anxiety state. Under indicated therapy her general condition improved, without any change in the laryngeal findings.

In October 1949, a King operation was carried out on the left side. During this operation the arytenoid cartilage broke in two, and the vocal process was anchored to the thyroid ala. At the close of the operation the glottic rima was about 5 mm. wide, but subsequent examinations indicated a narrowing of the airway to about 2 mm., probably the result of a broken suture. As a result, the patient was able to close her trache-

otomy cannula at rest and during mild exercise, but was unable to return to her normal activities. She had gained 10 pounds and had lost some of her nervousness.

On Nov. 28 the patient again entered St. Francis Hospital, and a complete arytenoidectomy was performed through Seiffert's self-retaining laryngoscope, as described above. In order to preserve the vibrations of the intact right cord, it was decided to operate again on the left side, which had already been subjected to the above mentioned operation by the extralaryngeal approach. At this time the remaining portion of the arytenoid cartilage was removed by submucous resection. The scar tissue resulting from the previous operation was divided to permit further lateral motion of the left cord. The curved electrocautery tip was then inserted repeatedly along the posterior one-half of the left vocal cord, and the desired lateral fixation was obtained. Great care was taken in inserting the acrylic obturator into the newly formed triagular opening at the posterior end of the larynx.

The postoperative course of this patient was entirely uneventful. The small amount of edema began to subside on the third postoperative day, and the patient was permitted to get up on the same day. The acrylic obturator was left in position for a total of five days. The patient was dismissed from the hospital on the tenth postoperative day. She was again hospitalized on Jan. 24, 1950, when the tracheotomy stoma was closed under local anesthesia.

At the present time, the resulting glottic opening, as viewed indirectly, is 4 mm. wide posteriorly. Shortness of breath on exertion and the inspiratory stridor have completely disappeared. The patient is able to lead a normal existence, and has returned to her former position as a secretary. Her voice showed little change after the arytenoidectomy; it is hoarse, but adequate for all purposes, including prolonged telephone conversations. She has had a further 10 pound gain in weight and has lost her anxiety.

CONCLUSION.

I have found the intralaryngeal approach for surgical correction of bilateral abductor paralysis most satisfactory. I fully agree with Thornell[21] that "arytenoidectomy from within the larynx has been found feasible and not particularly difficult for one trained in the use of the suspension apparatus." Modification of the Thornell operation by the adaptation of the self-retaining laryngoscope and additional dissecting instruments was intended to simplify the procedure and increase its popularity. It is my firm belief that laryngologists trained in the use of the suspension or self-retaining laryngoscope will prefer this intralaryngeal approach. "For those surgeons without such training and equipment, the King operation or one of its modifications will and should continue to be the operation of choice."

BIBLIOGRAPHY.

1. Baker, C. H.: Report of a Case of Abductor Paralysis with Removal of One Vocal Cord. *Jour. Mich. Med. Soc.*, 15:485, Oct., 1916.

2. Hoover, W. B.: Bilateral Abductor Paralysis; Operative Treatment by Submucous Resection of the Vocal Cords. *Arch. Otolaryngol.*, 15:339-355, Mar., 1932

3. Lore, J. M.: A Suggestive Operative Procedure for the Relief of Stenosis in Double Abductor Paralysis: An Anatomic Study. *Ann. Otol., Rhinol. and Laryngol.*, 45:679-686, Sept., 1936.

4. King, B. T.: A New and Function-Restoring Operation for Bilateral Abductor Cord Paralysis. Preliminary Report. *Jour. A. M. A.*, 112:814-823, Mar. 4, 1939.

5. King, B. T.: A New and Function-Restoring Operation for Bilateral Abductor Cord Paralysis. *Trans. Am. Laryngol. Assn.*, 61:264-296, 1939.

6. King, B. T.: Progress in Dealing with Vocal Cord Paralysis. *Jour. Int. Coll. Surg.*, 12:288-296, May-June, 1949.

7. Morrison, L. F.: Further Observations on the King Operation for Bilateral Abductor Paralysis. *Ann. Otol., Rhinol. and Laryngol.*, 54:390, June, 1945.

8. Kelly, J. D.: Surgical Treatment of Bilateral Paralysis of the Abductor Muscles of the Larynx. *Trans. Am. Acad. Ophth.*, 45:133-145, 1940.

9. Kelly, J. D.: A Supplementary Report on Extralaryngeal Arytenoidectomy as a Relief for Bilateral Abductor Paralysis of the Larynx. *Ann. Otol., Rhinol. and Laryngol.*, 52:628-636, Sept., 1943.

10. Galloway, T. C.: Modified Kelly Operation for Abductor Paralysis. *Arch. Otolaryngol.*, 34:1197, Dec., 1941.

11. McCall, J. W., and Gardiner, F. S.: A Simplified Operation for Bilateral Abductor Paralysis. The Laryngoscope, 53:307-311, May, 1943.

12. Wright, E. S.: The Kelly Operation for Restoration of Laryngeal Function Following Bilateral Paralysis of the Vocal Cords; Report of Three Cases. *Ann. Otol., Rhinol. and Laryngol.*, 52:346-358, 1943.

13. Orton, H. B.: Surgical Approach for Arytenoidectomy in Bilateral Abductor Paralysis of the Larynx. The Laryngoscope, 53:709-716, Nov., 1943.

14. Orton, H. B.: Extralaryngeal Approach for Arytenoidectomy. Bilateral Abductor Paralysis of the Larynx. *Ann. Otol., Rhinol. and Laryngol.*, 53:302-303, June, 1944.

15. Trotter, Sir W.: A Method of Lateral Pharyngotomy for Exposure of Large Growth in the Epilaryngeal Region. *Jour. Otol., Rhinol. and Laryngol.*, 35:289, Oct., 1920.

16. Woodman, De G.: The Open Approach to Arytenoidectomy for Bilateral Abductor Paralysis, with a Report on 23 Cases. *Ann. Otol., Rhinol. and Laryngol.*, 57:695, Sept., 1948.

17. Kelly, J. D.: Some Problems in the Surgical Treatment of Bilateral Abductor Paralysis of the Larynx. *Ann. Otol., Rhinol. and Laryngol.*, 53:461-468, Sept., 1944.

18. Thornell, W. C.: Intralaryngeal Approach for Arytenoidectomy in Bilateral Abductor Paralysis of the Vocal Cords; Preliminary Report. *Arch. Otolaryngol.*, 47:505-508, Apr., 1948.

19. THORNELL, W. C.: Intralaryngeal Approach for Arytenoidectomy in Bilateral Abductor Vocal Cord Paralysis. *Trans. Am. Acad. Ophth.*, 53: 631-636, July, 1949.

20. THORNELL, W. C.: A New Intralaryngeal Approach in Arytenoidectomy in Bilateral Abductor Paralysis of the Vocal Cords: Report of Three Cases. *Arch. Otolaryngol.*, 50:634-639, Nov., 1949.

21. THORNELL, W. C.: Personal communication to the author.

22. HAVENS, F. Z.: Personal communication to the author.

23. KING, E. D.: Personal communication to the author.

KERATOSIS OF THE LARYNX. REPORT OF A CASE WITH UNDERLYING CARCINOMA IN SITU.

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In reviewing the literature on keratosis of the larynx, one finds surprisingly little has been written on this most important subject. Particularly is this true in the era before 1940. This is all the more surprising since this most important lesion carries with it the stigma of being precancerous. Fortunately in the past decade several excellent articles have been written by such authorities as Clerf, Graham, New, Erich and several others. The textbooks also pay scant attention to this condition and often dismiss the subject with one or two inadequate paragraphs. Adding to the difficulty is the fact that there appears to be a definite diversity of opinion among the laryngologists and pathologists as to the terminology to apply to the various hyperplastic changes of the epithelium found in the larynx. One often finds such terms as keratosis, hyperkeratosis, leucoplakia, pachydermia, laryngis, hyperkeratosis laryngis and others used synonymously by some, while others consider each a distinct and separate entity.

Clerf[1] states that keratosis, hyperkeratosis or leucoplakia is primarily a localized benign hyperplasia of the epithelium, which does not invade the submucosa. This epithelial hyperplasia of the larynx may assume varying gradations from that of a simple profuse thickening as seen in chronic inflammatory reactions to that of large papillomatous masses that may fill the entire larynx. The whitish surface, which appears smooth or granular or may exhibit elevations or villus projections, stands out in contrast to the surrounding cordal mucosa, which often is the seed of a chronic inflammatory process.

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New and Erich[2] differentiate epithelial hyperplasia from keratosis or leucoplakia in that the former consists of masses of piled up epithelial cells in contrast to the latter, where one finds a hyperkeratosis of the epithelium with a much thickened mucous membrane with epithelial down growths.

Pachydermia as considered by Hayes Martin[3] is a counterpart of the leucoplakia found commonly in the oral mucous membrane. The differential diagnosis of pachydermia from that of the other keratoses of the larynx is difficult and often impossible. In essence, he states that it is a response of these tissues to long standing chronic irritation and for the development of this lesion, the nature of the irritant is of less importance than its chronicity.

The term hyperkeratosis seems to describe most accurately the lesions discussed in its various gradations by the different authors.

These keratoses are rarely associated with similar changes in the oral mucous membrane as were seen in the case to be described.

Hayes Martin states that the percentage of intraoral keratosis or leucoplakia is extremely low in leucoplakia of the larynx or in the association with carcinoma of the larynx. He goes so far as to state that rarely is a case of leucoplakia of the larynx associated with oral leucoplakia.

Some factors stand out in all cases of keratosis of the larynx: *1.* there is a local keratinization of the epithelium without invasion of the submucosa. In discussing keratosis in general, Reiman considers it to be primarily a condition in which there is shedding of the superficial layers, the cells piling up and producing islands known as keratinized islands because of the excess keratin in the cells. *2.* In all forms of keratosis appearing on the vocal cord, there is one change commonly observed which has elicited the widest interest and speculation: it is the variable amount of inflammatory reaction noticed in the submucosal layer. This has caused much comment by Graham, New, and Erich and others as to its

prognostic importance. This inflammatory reaction is diffuse throughout the mucous membrane of the larynx and shows areas of intensity about the base of the individual lesion. Graham[4] remarks that the presence of this inflammatory reaction is of the utmost importance and that a long step forward in determining the underlying cause of the whole process will have been made when this phenomenon is explained.

ETIOLOGICAL FACTORS.

While the causes of keratosis appear still to be in the realm of theory, there are predisposing factors well established in the minds of most otolaryngologists. Outstanding among these is that of long standing irritation. This is in keeping with the observation that keratosis is frequently associated with laryngitis of long standing. Hayes Martin flatly states that keratosis of the upper respiratory tract is a response of the mucous membrane of this tract to long standing chronic irritation. He further evaluates this statement by saying that in the development of the lesion, the nature of the irritant is of less importance than its chronicity. The lesion is most commonly observed in male adults who are excessive smokers.

Virchow[5] noted its appearance in alcoholic addicts or excessive voice users such as street hawkers. On the other hand, Clerf does not feel that excessive use of the voice or alcoholism is important.

Much work has been done experimentally and clinically in regard to the disturbances of vitamin A metabolism as related to keratinization of the skin. Along this line, Wallbach and Howe[6] have reported changes in epithelial structures following the deprivation of fat soluble vitamin A in experimental studies on rats. In these experiments which have been borne out by clinical evidence, it is found that various parts of the respiratory tract will undergo a keratinization of the epithelial surface when vitamin A metabolism was disturbed. Graham[4] was able to report a most significant case of keratinization of the larynx in which a remarkable regression of the entire lesion was observed as soon as attention was given to

the vitamin intake in the patient's diet; this, despite a histological diagnosis of carcinoma in association with keratosis.

PRECANCEROUS LESION.

Many writers have applied the term precancerous lesion to this type of pathological change seen in the larynx. Where one finds decided hyperplasia of the epithelium of the larynx in response to long standing chronic inflammatory reaction, this hyperplasia frequently suggests early malignant changes. Crawford, Negus[6,7] and others have repeatedly remarked that localized or diffuse overproduction of the superficial epithelium offers considerable difficulty in deciding whether malignant changes are commencing in deeper layers of what is potentially a precancerous lesion. It is impossible in examination of such tissue to state categorically which ones will develop cancer and in which recovery will occur.

Morrison[8] reported a case of keratosis in which a carcinoma apparently developed as a single small island of malignant cells in the subepithelial layer beneath a mucus gland. New and Erich[2] have reported 10 cases, two of which developed a squamous cell carcinoma. In both cases the carcinoma was discovered only histologically. Clerf cites four cases of keratosis observed over a period varying from two to four years, which eventually developed carcinoma.

Coplin[9] appropriately stated that histology rarely, if ever, tells us what a cell is going to do before it does it, although many of us are deluded into the belief that we can deduce the future of a cell from a study of the past, present or both. Most authorities having concluded that chronic inflammatory changes in various forms often precede the appearance of malignancy, that keratosis of the larynx reveals just such a change, and that in most instances it has been definitely shown to have undergone malignant degeneration; it should, therefore, be considered a precancerous lesion of the utmost importance and treated as such, preferably by surgical removal.

Jackson,[10] in his book, Diseases and Injuries of the Larynx, states that leucoplakia of the larynx is definitely a precancerous lesion. It is impossible clinically to determine whether such a lesion has undergone malignant change in its early stages, and it often requires considerable histological study. In the broader sense, however, such a lesion presents a great potential danger and should be treated by surgical removal.

PATHOLOGY.

Grossly, one may find a simple profuse thickening or localized areas of thickening of the epithelial, appearing in the form of single or multiple chalky, white elevations on the surface and edge of one or both vocal cords. Such localized areas of thickening may be considered as small masses of hyperplastic squamous epithelium, which at times may suggest papillomas but lacking the usual characteristic arrangements. The basement membrane is usually regular but may push into the submucosa in small irregular projections. In most instances a good basal layer of epithelium is observed. In the submucosal layers changes of an inflammatory nature are seen with localized areas of intensity about the base of the individual lesion. There are present many cells of an inflammatory origin with numerous thin walled vessels, fibrosis and edema. Those lesions which show considerable inflammatory changes and great irregularity of the basement membrane should be dealt with immediately in the same manner as early carcinoma despite the lack of clinical or histological evidence of the presence of carcinomatous changes.

SYMPTOMS.

While the laryngoscopic picture may vary greatly from that of a diffuse infiltration to that of localized areas of thickening or even papillomatous changes, there is commonly just one symptom, mainly, a change in voice, ranging from a slight huskiness to a coarse hoarseness. No pain, dyspnea or allied laryngeal symptoms are observed until very late in the course of the disease, when it has progressed into the stage of carcinoma.

TREATMENT.

There is no doubt that keratosis of the larynx frequently offers considerable difficulty in deciding whether or not malignant changes are already commencing in the deeper layers. Many otolaryngologists and pathologists declare it is impossible to determine that such a change has occurred other than by histological study, and even then a definite diagnosis is often difficult. This is aptly brought out by the two cases of Graham in which carcinoma was determined only through histological study. Coupled with this is the fact that one cannot prognosticate clinically or histologically whether any given case will or will not proceed into a condition of carcinoma. The consensus is that keratosis is a precancerous lesion and should be considered and treated as such. A progressive change in appearance or an increase in symptoms gives warning that a simple process, possibly present for years, may be changing its character. Certainly all cases warrant and demand histological study through biopsy even though this may not prove anything as demonstrated in Morrison's case and the case to be detailed here. In certain cases patients recover on a regimen of no smoking, vitamin therapy, vocal rest and removal of the keratotic material by various appropriate surgical measures. These measures are often inadequate and keratotic masses reappear despite frequent removal and even stripping of the cords as described by Putney, Clerf and Loré.[11,12]

Jackson and Jackson[10] state that the treatment of precancerous conditions when recognized is urgently called for. Keratosis requires removal, notwithstanding the fact that in many cases it may exist for years before developing malignant qualities. When the lesion's appearance is suggestive of carcinoma, then laryngofissure is definitely indicated, even though none may be found histologically. Close observation and repeated examination of all these lesions is imperative along with biopsy, so that one can detect the earliest possible changes in a lesion so predominantly precancerous and in which so much difficulty arises in our ability to determine even histologically if carcinoma is arising.

REPORT OF CASE.

J. W., male, white, age 36, first seen on Aug. 18, 1949. The reason for his visit was to arrange for a tonsillectomy while on vacation. During the course of history taking, it was noted that the patient spoke with some degree of hoarseness. On inquiry the patient stated that this symptom had been present in a varying degree from four to five years. He attributed this condition to his job in a foundry where he was subject to varying extremes of temperature and dampness. He smoked several packs of cigarettes daily and was a moderate drinker of alcohol.

Examination revealed this patient to have a rather diffuse leucoplakia of the buccal cavity on either side. Mirror examination of the larynx showed the right vocal cord in its middle third and extending slightly beyond this area toward the anterior commissure to be thickened, ragged, grayish-white and frayed. There seemed to be no distinct line of demarcation from the surrounding tissue. The remainder of the laryngeal mucosa and the left cord was entirely free of any suspicious change.

This patient was admitted to the hospital on Aug. 18, 1949, for direct laryngoscopy and biopsy.

General physical examination was essentially negative, laboratory studies within normal limits, Kahn negative.

Gross report of biopsy: "A piece of white, pink tissue measuring 0.25 by 0.2 cm., which is soft but not friable. Microscopic description: the tissue shows slight hyperkeratosis with parakeratosis. A thickened, stratified squamous epithelium and numerous cells of chronic inflammatory reaction in the subepithelial tissue. The epithelial cells are reasonably well arranged in regular fashion and there is no great variation in nuclear size. There are some mitotic figures seen. There is one area of stratified squamous epithelium where the cells are less regularly arranged and the demarcation between epithelium and connected tissue is not particularly sharp. One area shows apparently deeply situated epithelium, but this may well be merely the effect of a tangential cut. Base features suggest that this is a hyperkeratosis, but if there is the appearance of carcinoma on direct visualization of the region, I would suggest repeat biopsy."

The result of the biopsy and the type of lesion were discussed with the patient, and it was decided to proceed with a laryngofissure. This was done on Aug. 31, 1949. A tracheotomy was not performed.

Gross description of removed tissue: A mass of tissue measuring 2.5 by 1.5 by 0.5 cm. One surface is hemorrhagic, the other surface is yellow in color. There are firm nodular areas 0.5 cm. in diameter in this mass.

Microscopic description: It is noted that the stratified squamous epithelium is greatly thickened in areas with great tags of thin masses, both as to width and to depth, and seems to invade some of the tissue. There is much chronic inflammation also seen in the section. Several sections made in the tissue show that in one portion of this section the squamous epithelium sends large columns deep into the tissue, and in these particular column areas there appears to be an invasion about their bases. The nuclei of the cells here are enlarged, versicular looking, and occasional mitotic figures can be seen.

Diagnosis: Hyperkeratosis, squamous cell carcinoma of the larynx, grade I.

CONCLUSIONS.

Keratosis of the larynx is not a common disease. In association with oral leucoplakia it is rare.

Keratosis, while a relatively benign lesion, occurring chiefly in male adults, particularly in excessive smokers, carries with it the stigma of being a precancerous lesion.

While its etiology is unknown, experimental and clinical evidence tends to show that it may be related to some metabolic or food deficiency disease.

It is impossible, either clinically or histologically, to predetermine whether any given case will become cancerous. It is also frequently impossible to discover any malignant change other than through thorough biopsy or complete removal and serosections of the entire lesion.

By reason of their precancerous status, these cases should be kept under close observation and supervision. Any question or doubt as to the presence of malignant changes warrants the removal of the lesion by surgery. The status of leucoplakia is such as to lead many authorities to advocate surgery immediately; the surgery of choice is laryngofissure.

A case of hyperkeratosis of the larynx involving one cord in association with oral leucoplakia and exhibiting the presence of carcinoma determined by histological study is presented.

BIBLIOGRAPHY.

1. CLERF, L. H.: Keratosis of the Larynx. *J. A. M. A.*, 132:823-826, Dec. 7, 1946.

2. NEW, G. B., and ERICH, J. B.: Benign Tumors of the Larynx. A Study of 722 Cases. *Arch. Otol.*, 28:841, Dec., 1948.

3. MARTIN, HAYES: Nelson's Loose Leaf Surgery.

4. GRAHAM, H. B.: Keratosis of the Larynx. *Arch. Otol.*, 36:735, Nov., 1942.

5. VIRCHOW: Cellular Pathology.

6. WOLBACH, S. B., and HOWE, P. B.: Tissue Changes Following Deprivation of Fat Soluble Vitamin A. *Jour. Exper. Med.*, 42:735, 1925.

7. NEGUS, V. E.: Differential Diagnosis of Intrinsic Cancer of the Larynx. *Arch. Otol.*, 29:223, 1939.

8. Morrison, L. F.: Keratosis of the Larynx Showing Early Malignant Changes. *Trans. Pac. Coast Oto- and Ophthal. Soc.*, 25:235, 1940.

9. Coplin, W. M. L.: A Basis for Prevention of Cancer. *J. A. M. A.*, 78:1523, May 20, 1922.

10. Jackson and Jackson: Diseases and Injuries of the Larynx. Mcmillian Co., 1942.

11. Lore, J. M.: Stripping of the Vocal Cords. *Arch. Otol.*, 42:378, Dec., 1945.

12. Putney, F. J., and Clerf, L. H.: Treatment of Chronic Hypertrophic Laryngitis. *Arch. Otol.*, 31:525, June, 1940.

ADDITIONAL APPENDIX.

Cummings, George O.: Fibrosarcoma of the Larynx with Underlying Leucoplakia. *Ann. Otol., Rhinol and Laryngol.*, Vol. LV, June, 1948.

Jackson: Discussion on Precancerous Conditions of the Larynx. *Proc. Roy. Soc. Med.*

Klesdadt, W.: Hyperkeratosis and Precancerous Growths in the Larynx. *Ztschr.f. Hals-Nasen-u. Ohrenheilk.*, 19:500-504, 1928.

Pierce, N. H.: Leucoplakia Laryngis. *Ann. Otol., Rhinol. and Laryngol.*, Mar., 1920.

Padgett: Surgical Diseases of the Mouth.

NUTRITION AND THE EYE, EAR, NOSE AND THROAT.*

(With Excerpts from the Literature.)

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Diseases that afflict mankind seem to fall into two broad classifications — those from without and those from within — the infectious diseases from without and the degenerative diseases from within. Infections may attack swiftly; we often observe the cause and effect within weeks or even days. Degenerative disease may take a long time to develop. The lack of the needed nutrients in the cell shows no immediate effect; only after this lack has continued do its effects become evident — in caries of the teeth or degeneration of the structures around the teeth causing them to fall out, in chronic arthritis or degenerative changes in the heart and other organs. There is another contrast between infectious and degenerative diseases. Great advances have been made in the control of infections; but, in the civilized world degenerative diseases are on the increase. To be sure, nowadays people live longer than they used to; obviously the increase in degenerative disease is to that extent more apparent than real — entirely apart from any consideration of defective nutrition.

It seems that the laws of nutrition, when finally clarified, will lose much of their present complexity, as so well expressed by Harding:[1] Einstein has recently put forth his "Unified Field" theory which interlocks gravitation and electromagnetics in a single equation — aiming to explain more

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and more phenomena by the use of a single formula. In studying the phenomena of metabolism we have resorted to fractionation — dividing each problem into its tiniest parts. The method is good and we are acquiring tentative answers to a large number of research problems. Each solved problem is like an identified fragment of a jigsaw puzzle; the time is approaching when we can fit these together, and as we see the pattern emerge we can more readily see what the fragments begin to mean.

Early in our studies of Nutrition it seemed that the internist was the one best fitted to observe and study the effects of deficiencies; but gradually it became clear that the tissues most easily observed are the skin and the eye, ear, nose, mouth and throat.

It seems clear that degenerative disease is due to poor nourishment. This concept might be briefly expressed:—A lack of the right food produces pathologic change in all tissues. As to vitamins, the main lack in our national intake is B Complex. For thousands of years bread had been good food. Then millers began removing vitamins and minerals, leaving only impoverished flour. Grain products now furnish one third of our food — and ninety eight percent of flour is white. Refined sugar is also a carbohydrate from which vitamins and minerals have been extracted, and sugar accounts for actually one seventh of the total food calories of the United States. In general, foods which spoil are good and foods which do not spoil are bad.

From the ideal and also the practical standpoint it is discouraging to face the fact that civilization affords foods of poor quality. It is so different from the days when vegetables came direct from the garden, milk direct from the cow, meats direct from "the killing". In those days the boy took the grain to the mill, bringing home fresh-ground meal and flour. Today's predicament is the unavailability of such foods; the only possibility is to make the best of a bad situation.

In this vein, as to the impossible ideal, Lee[2] makes the following suggestions: Eat no white flour or refined sugar.

As sweets use only honey, brown sugar, maple sugar or refined saccharin; use *fresh-ground* wheat or other cereals exclusively, because some vitamins become oxidized in a few days. Freshness is the measure of wholesomeness. The very flavor of whole wheat changes day by day after it is ground up — like coffee; the finer the grinding, the faster its loss of vitamins. Many people are now finding the answer in a home grinder that can be obtained at low cost from department stores. Pasteurized milk is relatively valueless as a source of the calcium it is so highly recommended for, because pasteurization destroys the enzymes necessary for calcium assimilation; so rely on raw certified milk, if your local Medical Association makes it available. Citrus fruits are not dependable sources of "C" unless fresh. Oranges may be devoid of "C" if over three months old, although not differing in appearance from fresh fruit. Leafy vegetables may lose all their "C" in one week of storage, so the only dependable source is the roadside farmer or your own garden. Meat is kept in cold storage until it contains only a vestige of its original vitamin content; fresh fowl and fish are preferable for vitamin content. Peanut products, fresh roasted nuts or peanut butter, are good high protein substitutes for meat. Potatoes, like wheat, can support life indefinitely if eaten baked or boiled, but not mashed. Mashing exposes "C" to air causing its destruction. Potatoes have about one third the "C" content of oranges. Bananas contain every essential nutrient. So, we can list as basic dependable foods: milk, wheat, potatoes, bananas. Each will support human life if not devitalized by tampering. Meat will stand in this category, especially if fresh.

As long ago as 1906 Ladd and Stallings[3] showed that the only excuse for using bleach is that it makes flour whiter. Bleaching also makes inferior flour look as "good" as superior flour. Bleach injures gluten. Even then the question was raised whether the U.S. Government should not prohibit the bleaching process because of its degradation of the bakery products. Their conclusions, even in 1906, condemned the process of bleaching. Lee[4] comments that bleached flour was

declared in 1919 by the U.S. Supreme Court to be a threat to public health; however, the bleaching still continues. As to sugar — most people enjoy dessert; but Harding[5] points out that sweets make one feel well fed without being well fed.

At a recent Midwinter Convention of the Research Study Club we presented a simple method of finding out what a patient eats. This was printed and distributed among many — and adopted by some. It has been suggested that this be reproduced here.

* * * *

The nurse gives the patient a piece of paper on which there is an outline of "morning, noon and night" for two weeks. She then explains that immediately after each meal (while it is all fresh in mind) the patient is to write down what he ate; anything taken between meals is also to be written down. The patient reads his own handwriting to the doctor—at the end of two weeks. The doctor writes the word GOOD on one side of the page and BAD on the other side. He listens to the patient and interrupts as needed so that he can make notes under the heading GOOD or the heading BAD.

Analysis: You will feel a bit awkward for a while. Everyone does. However, you will be doing a great service to your patient if you simply have him cut down on everything that is made from white flour and sugar. As it were, they reduce the "high octane" of the fuel on which we travel. It is perfectly possible for our automobile to go along on a wretched gasoline — that is the trouble. We think that because we are getting along, everything is all right. Far from it. For health, human fuel requires a high-octane rating.

All of us are confronted daily with problems due, wholly or in part, to lack of the right foods. For example — a "stuffy" nose; non-suppurative sinuitis; lesions of the conjunctiva and cornea, angles of the mouth and tongue; beginning nerve deafness with no apparent cause; and "catching colds" frequently.

Before we used our present routine we wasted time collecting the information. Now we find that the whole procedure

can be done in twenty or thirty minutes, including instructions to the patient. The patient reads his own handwriting while you make notes. Then you give a piece of paper to the patient — on the one side the word BAD and on the other side the word GOOD; the patient then puts down your instructions in his own handwriting so that he also will have no difficulty in reading it.

Almost invariably you will find that there is a lack in the B Complex. Liver is prescribed twice weekly: oil is brought to a very high temperature in the skillet, the liver is dropped in until it is crisp on the outside — then turned over. The result is what we call "Eskimo Pie Liver" — crisp on the outside and rare on the inside. Wheat germ is helpful — a tablespoonful every morning or every other morning, given with cereal. Of course whole wheat bread, if you can get it. Large doses of the B Complex are often helpful; the amount may be reduced after the patient returns for further directions.

As to the sugar, exhort the patient to use as little as possible from now on. He may look shocked — until you explain that you have something far better—refined saccharin—five hundred times sweeter than sugar. It is perfect; no bitter taste. Naturally, saccharin cannot be dropped on any cereal, but instead is put in the milk or cream.

Well, so much for a beginning! Having once started you will then enlarge your information and be better and better able to answer specific questions — "Is this good?" — "Is that bad?" If this routine is followed the results will be fair, good or excellent. By prescribing the right food we are truly physicians of the eye, ear, nose and throat. If we do not, we are neglecting an important service to the patient.

* * * *

One disease concerning which our knowledge is by no means commensurate with our interest and concern, is poliomyelitis. The rhinologist has been concerned with this disease for a long time. It was first thought that the matter was quite simple — infection enters through the nose and attacks the central nervous system. Forty-one years ago a virus was found

in the degenerated areas of the nervous tissues; later it was thought that the cribriform plate was the portal of entry, and the zinc sulphate treatment came into vogue. The idea was to "toughen the mucosa" by spraying the zinc into the upper reaches of the nose. Although this treatment came to be used widely, it seemed so fantastic that we asked Dr. Leland Hunnicutt[6,7] to conduct experiments on dogs and monkeys for the Research Study Club. At that time the residents, internes and nurses of our County Hospital were all set to take this treatment. The histologic study by Dr. Hunnicutt showed a complete loss of olfactory epithelium and degeneration of the neurones leading from the surface. Because of these findings this treatment was abandoned at the hospital — and soon abandoned by everyone.

The opinion of authorities is well expressed by Bower.[8] Based upon his experience with 6000 cases in a four year period at the Los Angeles General Hospital Bower gives his up-to-date concepts of polio. The virus that causes this disease is the "Legio Debilitans". This virus occurs in three groups and representative strains from all three have been found. This disease of man has been reproduced by inoculating infectious human material into primates; and one strain has reproduced the disease in rodents. Whereas the virus may be recovered in only a low percentage of cases early in the disease by swabbing the nasopharynx, it may be obtained with relative ease and frequency for weeks and even months from stools — both of patients and of asymptomatic carriers. Bower states that in his hospital during this four year period doctors, nurses and attendants worked without masks yet none contracted the disease. (In contrast to this the 1934 epidemic had affected many nurses and several doctors in the same hospital.) During an epidemic people should be told to keep the hands clean constantly and to avoid excessive fatigue; no other steps have shown evidence of inhibiting the spread of the disease during an epidemic.

The National Foundation of Infantile Paralysis frankly states that we do not know how to prevent polio or how to stop it. The portal of entry is unknown. The usual methods

of combating infection — serums, vaccines and chemotherapy — have had no effect. Quarantine has been abandoned as useless.

A recent experience illustrates our helplessness. A few days ago a Davis Cup champion aged 21 appears at 11 A.M. — and his match is to be at 1 P.M. His throat is so "sore" that he can't swallow. He had been unable to eat any breakfast. No fever, pulse 60, blood-pressure normal and a perfectly normal appearance in nose, nasopharynx and throat. He is told "there is no infection, just a rheumatic sore throat, in the muscles"; he is advised to drink water in large quantities; to wear a light sweater, and play tennis. The next morning he is comfortable, and the next day entirely well. At the same time another young man also aged 21 presented a similar picture to a colleague; but the mother was the anxious kind, so for that reason and that reason alone, the patient was put in the hospital — where he died in 48 hours of bulbar polio — confirmed by autopsy. How can we prevent such things? What sort of disease is this polio?

It is no wonder that many strange and diverse suggestions have been advanced about this mysterious disease — all of them based upon what we eat. One group[9] suspects alkaloid poisoning from the milk of cows which have been forced by the hot dry season to eat Jimson weed and cocklebur. Another group of reports has just appeared in a popular magazine.[10] Several suspect that the virus is not the cause but the product of the disease; others note that areas with high content of calcium and phosphorus have little polio — and recommend eating bone-meal; another remarks the resemblance of polio to shell-fish disease and suggests this poisoning as a cause. Perhaps the most plausible speculation, although it has not worked out, was that lack of the B Complex, especially "B_1", leads to pathologic change in central nervous tissues. The theory advanced by several clinicians[11,12,13,14,15] is that the symptoms of polio resemble those of beri-beri which is recognized as due to lack of the B Complex, especially "B_1"; that "B_1" and "B_2" are concerned with cell respiration — which is most important in nerve tissue; that epidemics of polio were

unknown through the ages until one hundred years ago and that man's intake of "B_1" has been markedly reduced since the introduction about the same time of modern methods of milling wheat. These ideas are certainly of great interest and, at first thought, seem plausible. Local lesions due to the wrong food are observed in the eye, ear, nose and throat in patients otherwise apparently well and it seems reasonable that some such lesions could occur in the brain and cord. However, any nutritional disease like beri-beri and pellagra, continues on and on until the right food is given. In sharp contrast, polio often quits with the frost—as if cut off with a knife. Elaborate experiments with "B_1", by Elvehjem[16] and others[17] led to diametrically opposite findings: Mice deprived of "B_1" were actually *more* resistant to the polio virus. The outstanding point about polio is that it occurs in epidemics; this fact would seem to confirm it as a communicable disease.

* * * *

Looking backward, we come to realize more and more the value of the life work of Weston A. Price, D.D.S.[18] Three pioneers in Nutrition have been Dr. Price in the United States, Sir Robert McCarrison in India and the Cheshire Doctors in England. Dr. Price might well be called "The Charles Darwin of Nutrition." Turning from test tubes and microscopes to the hitherto unstudied evidence among human beings, Dr. Price sought the factors responsible for fine teeth among people who had them — the isolated primitives. The world became his laboratory. As he traveled, his findings led him to the belief that dental caries was merely one expression of physical degeneration from what he had suspected — nutritional deficiencies. Whole tribes and villages at varying latitudes, altitudes and temperatures provided many thousands of case studies which he carefully recorded by pen and camera. Specimens of the foods used by these isolated peoples were sent back to Cleveland, Ohio, for laboratory analyses—some of them over a period of years—to cover seasonal changes. Travelers, explorers and scientists have made it common knowledge that in their natural environment primitive peoples possess fine teeth. On this clue Dr. Price selected fourteen primitive groups, sufficiently isolated to have maintained them-

selves on their traditional diets. Many voyages carried him from villages in Switzerland and Gaelic villages in the Outer Hebrides to the Eskimos and Indians of North America, and to the Melanesian and Polynesian South Sea Islands. Thence he traveled to Africa, down to the Australian Aborigines, the New Zealand Maories, and back to the primitive Indians of South America in the high Andes and coastal plains. Wherever he went, Dr. Price found the same thing. Stalwart bodies were typical of primitives on their traditional diets — rich in essential food factors. Primitive peoples, no matter where located, who were successfully maintaining their tribal pattern from one generation to another were found to be living on highly nutritious foods. While tribal foods varied as to source and kind, each tribe had learned the foods essential to vitality and reproduction. Laboratory analysis of these foods showed them to be exceedingly high in proteins, vitamins, minerals and fat-soluble factors. In cranial and dental development contrasts are striking between primitive individuals and their modern counterparts living on "civilized" foods. Dr. Price noted changes in pattern — elongated facial structure, deformed dental arch and various other malformations. Well established racial patterns changed with the introduction of refined commercial foods; increasing impairments were observed in succeeding generations. The importance of nutrition during pregnancy has long been recognized, but Dr. Price's investigations showed that primitives understood and practiced preconception nutritional programs for both parents. Many tribes required a period of pre-marital nutrition and children were spaced to permit the mother to maintain full health and strength — thus fostering offspring of physical excellence. Foods of special value were rationed among the pregnant and lactating women, as well as the maturing boys and girls in preparation for future parenthood. These primitives, with their fine bodies, homogeneous reproduction, emotional stability and freedom from degenerative ills, stand forth in sharp contrast with those subsisting on the impoverished foods of civilization. This great contribution from a life's work has been available for many years — and yet its full meaning is only beginning to be appreciated.

Untimely death robbed us of the amazing Mickey.[19,20] He saw man as an integral part of the earth. The material part of man consists of a dollar's worth of chemicals which come from that thin layer of the earth's surface called the soil — except for the oxygen, nitrogen, hydrogen and carbon which come from its atmosphere. The soil upon which all life depends is a thin layer of an average depth of eight inches over the face of the land. The earth beneath is as dead and sterile as the moon. That thin film is all that stands between man and extinction. Not only that — deficient soil produces deficient men, animals and plants. When this mere film disappears man disappears—"and the place thereof shall know him no more". The Empire of Babylon was irrigated by the Tigris; the Tigris changed its course and Babylon became a desert. Vast stretches once forested and inhabited are now the Sahara.

In 1673 Archer[21] made statements which are pertinent today. He spoke of the great damage which comes upon most people by not knowing about their own "constitutions of body; in ignorance, they eat and drink simply to please the palate". "Many dig their graves with their teeth and cut off the thread of life sooner than is required by God or nature — besides living in sickness and disease". He reasons that each one should study his own food and thus be his own doctor. He sees the order of evolution in medicine — first healing; later prevention of illness; preservation of health comes first, restoration of health next. According to the old proverb that every man is a fool — or a physician — at forty, each should study "the friendly agreement or antipathy of food to his own body; and know the nature of all foods". Man is to be pitied who knows not the nature of what he eats. "Negligence in so necessary a knowledge has occasioned much sickness to many, and death to not a few".

From the time of Hippocrates until 1830, as Elvehjem[22] states, it was assumed that all foods supplied only a single universal "nutrient". It was in 1830 that Prout analyzed food into fats, carbohydrates and proteins; and it was not until 1906 that Gowland Hopkins called attention to a "special fac-

tor," to which Casimir Funk gave the name "vitamin" in 1911.

The otolaryngologist or the dentist has unusual opportunities to detect evidences of nutritional deficiency in the course of routine inspections. In emphasizing this, Mann[23] lists those most frequently found. Glossitis is perhaps the most important. Patients frequently complain of burning, soreness and ulcers of the tongue and inability to tolerate hot, spicy foods. Red, hypertrophied papillae at the tip and sides of a swollen "pie-crust" tongue suggest early pellagra. If the tongue is slick and "gelatinous" in appearance it suggests more chronic pellagra, a "B_2" deficiency or an anemia. Glossitis is a cardinal sign of danger although it does not tell what specific trouble is present. Almost always nutritional deficiencies coexist; it is rare to find only a single deficiency. The color and tissue tone of the mucous membrane is also an important indicator. An oral mucosa which is fiery red, but not swollen or easily bleeding, indicates early pellagra. In chronic pellagra the mucosa is dark red and pyorrhea is frequent. In scurvy the gums are dark red, spongy and bleed easily. Fissures at the angle of the mouth suggest "B_2" deficiency. Such lesions start as pale areas, usually bilateral and symmetrical. The angles are progressively eroded and macerated until a triangular, wet, "angry-looking" lesion results.

In the Birmingham Clinic 1102 infants and children with uncomplicated deficiency diseases were studied by Spies, Ewing and Mann.[24] The mothers of many of these children had deficiency diseases — more pronounced during pregnancy and lactation. It seems probable that some of the infants began to develop deficiency disease in utero. In these infants and children a period of poor general health preceded the appearance of lesions of pellagra, beri-beri or riboflavin deficiency. A study of 200 of these cases showed that characteristic lesions disappeared promptly on giving the appropriate synthetic vitamin. The promptness of response in many was startling. An example—12 months old boy; his mother "got down to skin and bones", diarrhoea and sore mouth during the last two months of pregnancy; had always preferred carbohy-

drate food; in the two years preceding this child's birth (the youngest of seven) the family had had no milk, family diet being corn meal, grits, dry beans and salt pork. This baby was breast-fed and, since mother's milk supply was scanty, when he was three months old she began giving him grits and cornbread soaked in water. A week before admission to hospital diarrhoea started; emaciated, dehydrated and acutely ill. Urine showed no nicotinic acid. First two days he received 125 mg. of niacinamide daily in divided doses. At the end of 24 hours diarrhoea had decreased; in 48 hours it had ceased, appetite returned and he received liberal foods suitable to his age; discharged on the fifth hospital day. Niacinamide was continued for a month; at home his food remained poor but he ate more than prior to this illness. He continued gaining for eight months when the weight leveled off. This is a sample of the experience in these 200 cases. It should be noted that the therapeutic effects did not manifest themselves so quickly in the mothers as in the children.

To determine the effects of heat-treated foods upon growth and development, Pottenger[25] conducted experiments for ten years, utilizing nine hundred cats of which records were completed on six hundred. His attention was directed to the need for such studies by the mortality among cats which had been fed cooked meats. These cats withstood operations poorly despite good surgical technic. In time, more cats were available than could be fed on scraps from the kitchen; so raw meat scraps were purchased — including muscle, bone and viscera. This raw meat was fed to a similar lot of cats, and within a very short time these survived surgery; they were healthier and their offspring more vigorous. This contrast was so striking that systematic feeding experiments were undertaken. Careful clinical notes were kept; in the case of death, autopsy was performed, cataloguing gross and microscopic findings; calcium and phosphorus determinations were made; and at the end of ten years all remaining animals were autopsied. In the first experiments one group received raw meats 2/3, raw milk 1/3 and cod liver oil; another group received cooked meats 2/3, raw milk 1/3 and cod liver oil. Those receiving raw meat

reproduced normally, litters averaged five, lactation was normal, offspring were normal. Cats receiving cooked meat reproduced a heterogenous lot, each kitten differing from his litter-mates; abortion was common — about 25 per cent in the first generation up to 70 per cent in the second generation; dystocia and death in labor were frequent; kitten mortality was high, often due to failure of lactation; vermin and intestinal infestations were frequent. Of the cats on cooked meats many showed "bones as soft as rubber". Kittens of the third generation all died within six months, thereby terminating the strain. Another group of cats, first fed on raw meat, were switched to cooked meat for six months and then returned to raw meat. Kittens from this group showed some stigmata although mothers appeared to be in good health. Subsequent generations improved as long as they received raw meat. Their resistance to disease, greatly diminished on cooked meat, gradually increased upon return to raw meat. Cats fed on raw meat maintained normal malar and orbital arches, nasal structures, dental arches and dentition. Adult cats on cooked meat began to show abnormal conditions in the mouth in from 3 to 6 months — gingivitis, paradentosis, loss of teeth — but no caries. Others fed on raw milk were compared with those fed on pasteurized, evaporated and sweetened condensed milk. Those on raw meat flourished; those on the altered milks fared badly. In brief, it appears that raw meat and milk are better for cats than altered meat and milk.

VITAMINS.

A review by Evans[26] of the recently established and still unknown components of the B_2 Complex describes two new factors, — Pteroylglutamic Acid (PGA) and B_{12}. PGA has already given promise of value in nutritional macrocytic anemia; and in conjunction with B_{12}, in sprue. Isolated only recently, B_{12} appears of value in Addisonian pernicious anemia. It is the most powerful therapeutic agent known; one millionth of a gram provokes hematologic response in man. Seven other members of the B_2 Complex have already been established.

Two patients had pernicious anemia, two had nutritional macrocytic anemia, one had non-tropical sprue. After intramuscular injections of B_{12} Spies, Lopez and Milanes[27] observed positive hematologic response in the two cases of pernicious anemia; also in the two cases of nutritional macrocytic anemia and in the case of non-tropical sprue. All cases showed clinical improvement, including well-being, alertness, general strength and vigor and relief of soreness and burning of mouth and tongue.

The plasma level of "C" and the prothrombin time were studied in one hundred and four cases of epistaxis by Neivert, Engelberg and Pirk.[28] Only eleven showed normal "C" and prothrombin time. The level of "C" was subnormal in 68; of these, 35 also showed subnormal prothrombin time. In 25 the "C" level was normal but the prothrombin time was subnormal. Vitamin therapy was applied in 62 of the 93 who were deficient in "C", in prothrombin time, or in both. The clinical response could be determined only in 36, of whom 27 were improved — suggesting that "C" as well as "K" has a place in the management of nasal hemorrhage.

Although hemorrhages due to prothrombin deficiency can usually be adequately controlled by "K", Butt, Magath and Seldon[29] present evidence that such benefit does not occur in patients with severe liver damage.

MINERALS.

It is too early to know what place isotopes may come to occupy in medicine. It is not too early, however, to be aware of difficulties and dangers which beset the way. Light upon this subject is shed by studies of plants, bringing out the remarkable advantages and also disadvantages. It is obvious that isotopes are new and powerful tools, permitting investigations not previously possible. Russell[30] shows how isotopes are used in studying plant growth — especially how plants absorb and utilize the nutrients obtained from soil. Isotopes act as "tracers"—a word which explains exactly their use in biologic research. They are used as indicators to show the

behavior of normal elements; for example, if the biologist is working with radioactive phosphorus it is in order to investigate normal phosphorus. The Geiger counter is so sensitive that if one ounce of radioactive phosphorus were spread uniformly over the entire surface of the earth it would be possible to check whether the correct amount had been placed on every square inch. Isotopes can be detected by both the ear and the eye. Photographic plates are also sensitive and provide visual details as to the tracer's distribution. It is possible to measure the amount of nutrient absorbed in a few minutes by the tip of a root less than one-fourth inch long. The use of tracers is in its infancy; the first explorer's map of any region is never so precise as those of later observers. Careful thought must be given to possible damage. The isotope may affect the growth of the plant. To make matters worse, the plant may still appear healthy and yet be damaged. The isotope is a new tool, fascinating, of great potential value, but not without serious dangers.

With atomic energy man can create elements at will for use in medicine, as expressed by Lawrence.[31] Ordinary elements can be made radioactive — a good example is in the use of iodine in thyroid cancer. A new carbon has been produced — At.Wt.14 instead of 12; its course can be traced in the living body of plant, animal and man. We may learn why chlorophyll is the only agent in nature than can harness energy from the sun for creating the food on which we live. Carbon 14 may enable us to create food from simple substances, just as the plant does. Man, in fact, stands at the gateway of a new world — provided of course he does not commit suicide inadvertently.

In the light of recent advances Deuel[32] considers that the attitude toward the importance of fats should be changed. They are no longer to be considered as "optional", but rather as required food. In studies of the growth of rats it appeared that diets containing 20-30 per cent fat gave optimal results. It was also found that rats on high-fat intake could accomplish a greater amount of work than those on low-fat intake. The development of the isotope technic has made it possible

to follow the various intermediates of fatty acid oxidation through their several pathways.

Calcium is required by animal and human bodies in larger percentage than any other element; as Albrecht[33] expresses it calcium heads the list of strictly soil-borne elements required in the nourishment of living tissues. As rocks are broken down to form soil there is an increase of active calcium. When heavy rainfall occurs and the temperature rises there is calcium depletion. Lower forms of life — like microbes, and higher forms — like plants and animals, all are part of this calcium picture. The distributions of the different plants and animals take their patterns very much according to the calcium supply.

BIBLIOGRAPHY.

1. HARDING, J. C. ALMY, D.D.S.: Lecture before Midwinter Convention, Research Study Club, Los Angeles, January 1950.

2. LEE, ROYAL, D.D.S.: Lee Foundation for Nutritional Research, Milwaukee 3, Wisconsin.

3. LADD, E. F., and STALLINGS, R. E.: "Bleaching of Flour". North Dakota Agricultural College, Government Station Bulletin, No. 72. Nov. 1906.

4. LEE, ROYAL, D.D.S.: Foundation for Nutritional Research. Milwaukee, Wisconsin, August 1947.

5. HARDING, J. C. ALMY, D.D.S.: Editorial, *Jour. Amer. Acad. Applied Nutrition*, Autumn 1948, p. 177.

6. HUNNICUTT, LELAND G.: "The Effects of Zinc Sulphate on the Olfactory Mucous Membrane in Dogs and Monkeys". Transactions, 27th Annual Meeting, Pac. Coast Oto-Ophthalmological Soc.

7. HUNNICUTT, LELAND G.: "The Effect of Drugs on the Nasal Mucous Membrane". Transactions, 29th Annual Meeting, Pac. Coast Oto-Ophthalmological Soc.

8. BOWER, ALBERT G.: "A Concept of Poliomyelitis". *Northwest Medicine*, February, March and April 1950.

9. BROMFIELD, LOUIS: Book — "Out of the Earth". Harper and Bros., New York.

10. Magazine—"Prevention". Rodale Press, Emmaus, Pa. June 1950, pp. 2-80.

11. HUFFMAN, O. V.: "The Etiology of Epidemic Poliomyelitis and Its Possible Relation to Beri-beri". *Med. Record*, June 17, 1911.

12. LEE, ROYAL, D.D.S.: Lee Foundation for Nutritional Research. Milwaukee, Wisconsin.

13. HELMS, KAREN: "Acute Anterior Poliomyelitis and Vitamin B Deficiency". *Med. Jour .of Australia*, Vol 1—28th year, pp. 717-722.

14. McCormick, Wm. J.: "Poliomyelitis. Infectious or Metabolic". *Archiv. Pediatrics*, Vol. 67, No. 2, Feb. 1950.

15. Odom, Wm. P., D.D.S.: "The Nutritional Aspects of Poliomyelitis". *Jour. Amer. Acad. Applied Nutrition*, Vol 2, No. 3, Autumn 1948.

16. Rasmussen, A. F., Jr.; Waisman, H. A.; Elvehjem, C. A.; and Clark, P. F.: "Influence of the Level of Thiamine Intake on the Susceptibility of Mice to Poliomyelitis Virus". *Jour. Infect. Dis.*, Vol. 74, 1944, pp. 41-47.

17. Schneider, H. A.: "Nutrition and Resistance to Infection". Book. Vitamins and Hormones. Academic Press, New York, pp. 35-70.

18. Price, Weston A., D.D.S.: A book, "Nutrition and Physical Degeneration". Amer. Acad. of Applied Nutrition, 1105 So. LaBrea, Los Angeles 19.

19. Mickey, Karl B.: Book. "Man and the Soil". International Harvester Co., Chicago, 1945, p. 35.

20. Mickey, Karl B.: Book, "Health from the Ground Up". International Harvester Co., Chicago, 1946.

21. Archer, John: Book. "Every Man His Own Doctor". Published by the author. London, 1673.

22. Elvehjem, C. A.: "Recent Progress in Nutrition and Its Relation to Drug Therapy". *J. A. M. A.*, April 3, 1948, Vol. 136, p. 14.

23. Mann, Arvin W., D.D.S.: Lecture before So. Calif. Dental Assn., Los Angeles, April 1948.

24. Spies, Tom D.; Ewing, G. D.; and Mann, A. W.: "Effect of Synthetic Niacinamide, Synthetic Thiamine and Synthetic Riboflavin on Infants and Young Children with Deficiency Diseases". *Archives of Pediatrics*, 61, 517-531, October 1944.

25. Pottenger, Francis M.: "The Effect of Heat-Processed Foods and Metabolized Vitamin D Milk on the Dentofacial Structures of Experimental Animals". *American Journal of Orthodontics and Oral Surgery*, Vol. 32, No. 8, pp. 467-485, August 1946.

26. Evans, Herbert M.: "The Recently Established and Still Unknown Components of Vitamin B_2". Presented at Midwinter Convention of Research Study Club. Los Angeles, Jan. 1949.

27. Spies, T. D.; Lopez, C. G., and Milanes, F.: "Observations on Hemapoietic Response of Persons with Tropical Sprue to Vitamin B_{12}". *Southern Medical Journal*, June 1948, 41:p. 523.

28. Neivert, Harry, M.D.; Engleberg, Recha, Ph.D.; Pirk, Leo A., Ph.D.: "Nasal Hemorrhage". *Arch. Otolaryngology*, Jan. 1948, pp. 37-45.

29. Butt, Hugh, M.D.; Magath, Thos. B., M.D., and Seldon, Thos. H., M.D.: "Hypoprothrombinemia". *Arch. Intern. Medicine*, Feb. 1948, pp. 131-136.

30. Russell, R. Scott: "Tracers in Plant Growth". Magazine — The Countryman. Winter 1949, Vol. XL, No. 2, p. 295. Burford, Oxford, England.

31. Lawrence, Wm. L.: Book. "Information Please Almanac". Doubleday and Co., Garden City, N. Y., p. 12.

32. Deuell, Harry J., Jr.: "Lipid Metabolism". *California Medicine*, Vol. 72, No. 4, April 1950.

33. Albrecht, Wm. A.: "The Land". Vol. 111, No. 1, Dec. 1943.

BOOK REVIEW.

Facial Paralysis. Being a Treatise on a Clinical Classification of Paralysis of the Facial Nerve. By J. Parkes Findlay, M.B., Ch.M., Honorary Assistant Aural Surgeon, Sydney Hospital. 47 pages with index and illustrations. Sydney, Australia: Angus & Robertson, 89 Castlereagh Street. 1950. Price 17/6.

This is a most interesting and useful monograph. The author has reviewed the anatomy of the facial nerve and its distributions. His comments on the history-taking and examination of the patient are very valuable.

The classification of facial paralysis which is offered is interesting: it is doubtful whether it will be quite generally accepted. One finds it difficult to agree, for instance, that the facial paralysis of sudden and complete onset is always accompanied by pain or, vice versa, that painless paralysis necessarily comes on gradually. It is difficult for this reviewer to agree that the differential diagnosis as presented by the author is entirely tenable. It is doubtful whether many will agree with the author in his dictum that "the treatment of vascular occlusion facial paralysis is surgical" and that "there is no clinical reason to delay the decompression of the nerve."

Whether the reader agrees with him or not, Dr. Findlay has presented a controversial subject in an interesting and provocative manner and the book is recommended to all interested in the facial nerve. T. E. W.

In Memoriam

SAMUEL J. KOPETZKY, M.D.,

1876-1950.

As we may be sure he would have wished it, Sam Kopetzky carried on his work with stimulating and tireless ardor up to the day of his death, Nov. 13, 1950.

Born in New York City in 1876, Samuel J. Kopetzky graduated from the College of Physicians and Surgeons, Columbia University, in 1898 and later from the Royal University Clinic, Berlin.

Sam was a true and fervent patriot; he served in the Army Medical Corps in the Spanish-American War and both World Wars, retiring with the rank of colonel. He was decorated with the Legion of Merit and the Silver Star.

Since 1939 he had been director of his department at the Polyclinic Hospital, professor of otolaryngology since 1920, on the staff of the Manhattan Eye, Ear and Throat Hospital for many years, consultant otologist at Beth Israel, Newark Beth Israel and Vassar Brothers (Poughkeepsie, N. Y.) Hospital. For his work on petrositis, France bestowed upon him the Order of Chevalier of the Legion of Honor. The American

Laryngological, Rhinological and Otological Society, Inc., of which he was president (1937), bestowed upon him its gold medal.

Dr. Samuel J. Kopetzky was active in the work of all the medical societies to which he belonged, and he became president of the medical society of the County of New York and later of the medical society of the State of New York. At the time of his death he was a Fellow of the American Medical Association, the New York Academy of Medicine, the American College of Surgeons, the American College of Ophthalmology and Otolaryngology.

A facile and fluent contributor to the otological literature, Dr. Kopetzky was one of the founders and, from 1935 to 1940, was the editor of *Medical Week*. He was literary chairman of the *New York State Journal of Medicine*. At the time of his death he was president of the Metropolitan Chapter of the Association of Military Surgeons of the United States. Several textbooks bear his name.

Our memories of Sam Kopetzky will contain as his outstanding traits kindness, understanding, a stable though tolerant and fair judgment, a friendship unaffected by differences of opinion, a seeker after the truth, a soldier and a gentleman.

To his widow, Mrs. Anah Doob Kopetzky, his son, Karl A., his daughter, Mrs. Yvonne K. Sterling, his three sisters and his many friends, our sincere sympathy in their great loss.

E. P. F.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

As of April 1, 1950.

Aurex Model F and Model H.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac; Beltone Harmony Mono-Pac; Beltone Symphonette; Beltone Mono-Pac Model M.
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago 16, Ill.

Dysonic Model 1.
Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.
Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Atomeer; Maico UE-Atomeer; Maico Quiet Ear Models G and H.
Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears Aurophone Model 200; 1947—Mears Aurophone Model 98.
Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver); Micronic Model 303. (See Silver Micronic.)
Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-4 Audiomatic; Microtone T-5 Audiomatic; Microtone Classic Model T9; Microtone Model 45.

Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model C; National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-1; Otarion Model E-1S; Otarion Model E-2; tarion Model E-4; Otarion Models F-1 and F-2.

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Models VH and VL (Standard); Paravox Model XT (Xtra-Thin); Paravox Model XTS (Xtra-Thin); Paravox Model Y (YC and YC-7) (Veri-Small).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55; Radioear Model 62 Starlet.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Silver Micronic; Silver Micronic (Magnetic and Crystal) Models 202M and 202C. (See Micronic.)

Manufacturer: Micronic Corp., 101 Tremont St., Boston 8, Mass.

(See Micronic.)

Silvertone Model 103BM.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Distributor: Sears-Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Silvertone Model M-35.

Manufacturer: Microme Co., 727 Atlantic Ave., Boston 11, Mass.

Distributor: Sears-Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Silvertone Model P-15.

Manufacturer: W. E. Johnston Mfg. Co., 708W. 40th St., Minneapolis, Minn.

Distributor: Sears-Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 600; Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.
Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Televox Model E.
Manufacturer: Televox Mfg. Co., 117 S. Broad St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 1700.
Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonemaster Model Royal.
Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill.

Trimm Vacuum Tube No. 300.
Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Model 63; Western Electric Model 64; Western Electric Models 65 and 66.
Manufacturer: Western Electric Co., Inc., 120 Broadway, New York 5, N. Y.

Zenith Model 75; Zenith Miniature 75.
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Aurex (Semi-Portable)—*Jour. A. M. A.*, 109:585 (Aug. 21), 1937.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago (10), Ill.

Precision Table Hearing Aid—*Jour. A. M. A.*, 139:785-786 (Mar. 19), 1949.
Manufacturer: Precision Electronics Co., 850 West Oakdale Ave., Chicago 14, Ill.

Sonotone Professional Table Set Model 50—*Jour. A. M. A.*, 141:658 (Nov. 15), 1949.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

AMERICAN OTOLOGICAL SOCIETY.

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Meeting: The Green Brier, White Sulphur Springs, W. Va., May 11-12, 1951.

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Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2, Pa.
Meeting: Green Briar Hotel, White Sulphur Springs, W. Va., May 9-10, 1951.

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Secretary: Dr. C. Stewart Nash, 108 Medical Arts Bldg., Rochester, N. Y.
Meeting: White Sulphur Springs, W. Va., May 6-8, 1951.

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Middle Section: Cleveland, Ohio, Jan. 15, 1951.
Southern Section: Miami, Fla., Jan. 17, 1951.
Western Section: San Francisco, Calif., Jan. 27-28, 1951.

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Richmond, Va., May 1-4, 1951.

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Place: Army and Navy Club, Washington, D. C.

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Meeting: Havana, Cuba, 1952. For information address the Secretary.

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Meeting: Havana, Cuba, 1952. For information, address Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa.

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Meeting: White Sulphur Springs, W. Va., May 7-8, 1951, P. M.

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Secretary of Section on Ophthalmology: Dr. Carroll McCoy.

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Secretary of Section on Otolaryngology: Dr. Edwin Scobee.

Place: Los Angeles County Medical Association Building, 1925 Wilshire Blvd., Los Angeles, Calif.

Time: 6:00 P.M., fourth Monday of each month from September to May, inclusive.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

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